



Case report on chronic liver disease with portal hypertension with massive ascites

Eunice Pala¹, N.Navya Anusha¹, J.Bhargava Narendra².

¹ Department of Pharmacy Practice (Pharm D 4/6) Andhra University College of Pharmaceutical Sciences, Vishakapatnam, Andhra Pradesh, India.

² Assistant Professor, Department of Pharmacy Practice, QIS College of Pharmacy, Vengamuapalem, Ongole, Andhra Pradesh, India.

ABSTRACT

Portal hypertension is an increase in the blood pressure within the system of veins called the Hepatic portal venous system. Veins coming from the stomach, intestine, spleen, and pancreas merge into the portal vein, which then branches into smaller vessels and travels through the liver. If the vessels in the liver are blocked due to liver damage, blood cannot flow properly through the liver. As a result, high pressure in the portal system develops. This increased pressure in the portal vein may lead to the development of large, swollen veins (varices) within the esophagus, stomach, rectum, or umbilical area (belly button). Varices can rupture and bleed, resulting in potentially life-threatening complications. We present a case of portal hypertension with massive ascites- A 43-year-old female was admitted with chief complaints of abdominal distension, abdominal pain, and shortness of breath prior to 15 days of presentation and the symptoms were gradually progressive in nature. Her past medical history reveals she had similar complaints in the past and is a known case of chronic liver disease. After admission, she has been diagnosed with portal hypertension along with Refractory ascites. The management included antihypertensives and human albumin. A high protein diet with salt and fluid restrictions, large-volume paracentesis procedure was carried out daily.

Key words:

Portal hypertension,
liver, Varices, Cirrhosis
Refractory ascites.

Article History:

Received On: 04.06.2020
Revised On: 22.07.2020
Accepted On: 25.07.2020

*Corresponding Author

Name: J.Bhargava Narendra
Email: jbn520@gmail.com
DOI: <https://doi.org/10.37022/wjcmpr.vi.153>

This article is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License. Copyright © 2020 Author(s) retain the copyright of this article.



INTRODUCTION

Cirrhosis of the liver refers to scarring of the liver which results in abnormal liver function as a consequence of chronic (long-term) liver injury. Cirrhosis occurs when the normal structure of the liver is disrupted by bands of scar tissue. One of the normal functions of the liver is to filter blood returning to the heart from the digestive system. In this cirrhosis condition, the presence of scar tissue causes increased resistance to blood flow through the liver, which leads to the development of high pressure in the veins that drain blood into the liver, resulting in a condition called 'portal hypertension' [3]. Portal hypertension can lead to life-threatening hemorrhage, ascites, and encephalopathy. Management of portal hypertension and its attendant complications, requires a multidisciplinary approach combining medical and endoscopic management, surgical or nonsurgical portosystemic shunting, and in some cases liver transplantation.

CASE REPORT

A 43 year old female patient was admitted in the general medicine department of King George Hospital with chief complaints of abdominal distension, abdominal pain and shortness of breath since 15 days. Her symptoms started 15 days prior to presentation and were gradually progressive in

nature. History of Shortness of breath grade III which increased on walking and was relieved in sitting position. Patient is a known case of Chronic Liver Disease and is under medication. There is a history of blood transfusion one month back and Pedal oedema since 15 days which was gradual in onset and progressive in nature and decreased urine output since 15 days and also decreased sleep and appetite. The patient is not a known smoker or alcoholic. She takes a mixed diet. History of yellowish discoloration of urine, skin and sclera. There is a history of similar complaints in the past and was admitted in the hospital twice. Patient has no history of hypertension, Diabetes mellitus, coronary artery disease, cardiovascular accident, tuberculosis, epilepsy, thyroid disorders, or surgeries. No similar complaints in the family. On examination the patient was conscious and coherent. History of pedal oedema. There is no history of cyanosis. There is no history of clubbing. There is no history of lymphadenopathy. Patient was pallor along with an icterus and edema. Her pulse was 100 beats per minute and blood pressure was 100/160 mmHg. On systematic examination distended abdomen was observed with ascites, dilated veins and positive fluid thrill. On Cardiovascular examination normal S1 and S2 sounds. No murmurs.

LABORATORY REPORTS INCLUDE

CBC: Hb-8mg/dl; TC-3400 lakhs/mm³, DC-P80/L20;
ESR-14mm/hr; CUE: albumin(-); sugar(-);

Case Report

microcells-(pus cells(2)); LFT:tbil-0.6;inj INBIL-0.2;sgpt-48u/l;sgot-72u/l;alp-188; Electrolytes:Na-136;K-3.34;Cl-107;RFT:urea-86mg/dl; Creatinine-4mg/dl.

Patient was treated with Inj.Lasix 20mg IV BD;Tab. Aldactone 50mg BD, Inj.Albumin 100ml IV OD,Inj.Ceftriaxone 1g IV BD,Inj.Pantoprazole 40mg IV OD,Syrup Lactulose 10ml TID ,Inj.Vitamin K S/C,High protein diet was recommended with salt and fluid restriction.After one day of admission ,Large volume paracentesis (2lit) was extracted and Tab.ciprofloxacin 500mg BD ,Tab.Metrogyl 400mg TID were added into medication.I/O charting was done.Patient was diagnosed as a known case of chronic liver disease with portal hypertension with Refractory Ascites.

DISCUSSION

The most common cause of portal hypertension is cirrhosis of the liver. Cirrhosis is scarring which accompanies the healing of liver injury caused by hepatitis, alcohol, or other less common causes of liver damage [4]. In cirrhosis, the scar tissue blocks the flow of blood through the liver. Other causes of portal hypertension include blood clots in the portal vein, blockages of the veins that carry the blood from the liver to the heart, a parasitic infection called schistosomiasis, and focal nodular hyperplasia, a disease seen in people infected with HIV, the virus that may lead to AIDS. Sometimes the cause is unknown,like in this case. The onset of portal hypertension may not always be associated with specific symptoms that identify what is happening in the liver. But if you have liver disease that leads to cirrhosis, the chance of developing portal hypertension is high.

The main symptoms and complications of portal hypertension include:Gastrointestinal bleeding marked by black, tarry stools or blood in the stools, or vomiting of blood due to the spontaneous rupture and hemorrhage from varices, Ascites (an accumulation of fluid in the abdomen),Encephalopathy or confusion and forgetfulness caused by poor liver function,Reduced levels of platelets, blood cells that help form blood clots, or white blood cells, the cells that fight infection [4].

diagnosis of portal hypertension sometimes can be based on the presence of ascites or of dilated veins or varices as seen during a physical exam of the abdomen or the anus. Various lab tests, X-ray tests, and endoscopic exams may also be used.Here , UGI endoscopy is used whereby coming to a conclusion where there is massive ascites with B/L grade I/II, and there is further decrease in renal parenchyma.The prognosis for patients with ascites due to liver disease depends on the underlying disorder, the degree of reversibility of a given disease process, and the response to treatment.

most causes of portal hypertension cannot be treated. Instead, treatment focuses on preventing or managing the complications, especially the bleeding from the varices. Diet, medications, endoscopic therapy, surgery, and radiology procedures all have a role in treating or preventing the complications. Other treatment depends on the severity of the symptoms and on how well your liver is functioning.

Endoscopic therapy. This is usually the first line of treatment for variceal bleeding and consists of either banding or

sclerotherapy. Banding is a procedure in which a gastroenterologist uses rubber bands to block off the blood vessel to stop bleeding. Sclerotherapy is occasionally used when banding cannot be used and is a procedure in which a blood-clotting solution is injected into the bleeding varices to stop bleeding. Medications-Nonselective beta-blockers (nadolol or propranolol) may be prescribed alone or in combination with endoscopic therapy to reduce the pressure in varices and further reduce the risk of bleeding. Nonselective beta blockers are also prescribed to prevent a first variceal hemorrhage in a patient with varices that are felt to be at risk for bleeding. Esophageal variceal banding has also been used for that purpose, especially in patients who can't take beta blockers. The drug lactulose can help treat confusion and other mental changes associated with encephalopathy.

Ambulatory patients with an episode of cirrhotic ascites have a 3-year mortality rate of 50%. The development of refractory ascites carries a poor prognosis, with a 1-year survival rate of less than 50% [2]. Sodium restriction (20-30 mEq/d) and diuretic therapy constitute the standard medical management for ascites and are effective in approximately 95% of patients. Water restriction is used only if persistent hyponatremia is present. More recent research has focused on the treatment of refractory ascites with aquaretics—vasopressin V2-receptor antagonists that promote excretion of electrolyte-free water and thus might be beneficial in patients with ascites and hyponatremia [9]. Although study results have been promising(5) ,aquaretics still await approval by the Food and Drug Administration (FDA). In a multicenter study that assessed the safety and efficacy of an automated pump system for the treatment of refractory ascites in 40 patients at 9 centers, Bellot et al reported the automated pump was an efficacious tool to remove ascites from the peritoneal cavity to the bladder. During the 6-month follow-up period, 90% of the ascites was removed with the pump system; there was also a significant reduction in the monthly median number of large volume paracentesis as well as a reduction in the number of cirrhosis-related adverse events [6]. Therapeutic paracentesis may be performed in patients who require rapid symptomatic relief for refractory or tense ascites. When small volumes of ascitic fluid are removed, saline alone is an effective plasma expander [7].The removal of 5 L of fluid or more is considered large-volume paracentesis [1] .Total paracentesis, that is, removal of all ascites (even >20 L), can usually be performed safely. Supplementing 5 g of albumin per each liter over 5 L of ascitic fluid removed decreases complications of paracentesis, such as electrolyte imbalances and increases in serum creatinine levels secondary to large shifts of intravascular volume. Note: The AASLD indicates that post paracentesis albumin infusion may not be necessary for a single paracentesis of less than 4 to 5 L ((class I, level C recommendation); however, for large-volume paracentesis, an albumin infusion of 6-8 g per liter of fluid removed appears to improve survival and is recommended (class IIa, level C recommendation) [8].

Patients can actually be maintained free of ascites if sodium intake is limited to 10 mmol/d. However, this is not practical

outside a metabolic ward. Twenty-four-hour urinary sodium measurements are useful in patients with ascites related to portal hypertension in order to assess the degree of sodium

Case Report

anorexia, monitor the response to diuretics, and assess compliance with diet. For grade 3 or 4 ascites, therapeutic paracentesis may be necessary intermittently [10].

Sodium restriction of 500 mg/d (22 mmol/d) is feasible in a hospital setting; however, it is unrealistic in most outpatient settings. A more appropriate sodium restriction is 2000 mg/d (88 mmol). Indiscriminate fluid restriction is inappropriate. Fluids need not be restricted unless the serum sodium level drops below 120 mmol/L. (10) When the patient is admitted she was treated with injection Lasix 20mg IV BD (if BP is greater than 100/60 mmHg), and other Tablet Aldactone 50mg BD, injection ceftriaxone 1g IV BD, injection pantoprazole 40mg IV BD, injection 25% dextrose IV TID, injection Human albumin 100ml IV, injection albumin 20% 1 ampoule IV OD, syrup Lactulose.

On further diagnosis where the known case of decompensated CLD with portal hypertension and hepatorenal syndrome the patient was administered with injection vitamin K (subcutaneous). Input and output charting was monitored. Tablet ciprofloxacin 500mg BD and tablet metrogyl 400mg TID were also added. After a couple of days the patient was examined with constipation for which bowel enema and proctolysis enema were given. After 7 days of initiation of therapy the symptoms appeared to subside. Maintaining good nutritional habits and keeping a healthy lifestyle may help in avoiding portal hypertension.

LONG TERM TREATMENT MONITORING

The best method of assessing the effectiveness of diuretic therapy is by monitoring body weight and urinary sodium levels. In general, the goal of diuretic treatment of ascites should be to achieve a weight loss of 300-500 g/d in patients without edema and 800-1000 g/d in patients with edema.

CONCLUSION

Chronic liver disease occurs when permanent structural changes within the liver develop secondary to long standing cell damage, with the consequent loss of normal liver architecture. Symptomatic treatment has been given in the present case and the aim of the treatment lies in mobilising the abnormal collection of third space fluid (intra abdominal fluid) and this is achieved by the combination of salt reduction and fluid restriction by about 1-1.5lt per day. Other treatments such as use of diuretics, paracentesis, Transjugular intrahepatic portosystemic shunting (TIPS), spontaneous bacterial peritonitis (SBP) and liver transplantation can be used. Since the patient is already a known case of chronic Liver disease, portal hypertension and ascites are the complications that arose as a result of chronic liver disease and should seek medical assistance in case of reappearance of symptoms. The most important aspect of patient education is determining when therapy is failing and recognizing the need to see a physician. Unfortunately, in most cases, liver failure has a dismal prognosis. All patients must be taught which complications are potentially fatal and the signs and symptoms that precede them.

REFERENCES

1. Pericleous M, Sarnowski A, Moore A, Fijten R, Zaman M. The clinical management of abdominal ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: a review of current guidelines and recommendations. *Eur J Gastroenterol Hepatol.* 2016 Mar. 28(3):e10-8. [Medline].
2. Fede G, D'Amico G, Arvaniti V, et al. Renal failure and cirrhosis: a systematic review of mortality and prognosis. *J Hepatol.* 2012 Apr. 56(4):810-8.
3. K Thapaliya, A Bhandary, S Bhanset and B Aryal: Clinical status of furosemide on Liver Cirrhosis with Portal Hypertension And Ascites. *Journal of Chitwan Medical college* 2013,3(3):65-66.
4. WebMD Medical Reference Reviewed by Minesh Khatri, MD on December 21, 2018. (<https://www.webmd.com/digestive-disorders/digestive-diseases-portal#1>).
5. Gines P, Wong F, Watson H, et al, for the HypoCAT Study Investigators. Effects of satorvastatin, a selective vasopressin V(2) receptor antagonist, on ascites and serum sodium in cirrhosis with hyponatremia: a randomized trial. *Hepatology.* 2008 Jul. 48(1):204-13. [Medline].
6. Bellot P, Welker MW, Soriano G, et al. Automated low flow pump system for the treatment of refractory ascites: a multi-center safety and efficacy study. *J Hepatol.* 2013 May. 58(5):922-7. [Medline].
7. Sola-Vera J, Minana J, Ricart E, et al. Randomized trial comparing albumin and saline in the prevention of paracentesis-induced circulatory dysfunction in cirrhotic patients with ascites. *Hepatology.* 2003 May. 37(5):1147-53. [Medline].
8. Runyon BA, American Association for the Study of Liver Diseases. Management of adult patients with ascites due to cirrhosis: update 2012. National Guideline Clearinghouse. Available at <http://guideline.gov/content.aspx?id=45103&search=ascites>. Accessed: Nov 6, 2014.
9. Gines P, Cardenas A. The management of ascites and hyponatremia in cirrhosis. *Semin Liver Dis.* 2008 Feb. 28(1):43-58. [Medline].
10. Medscape articles on Ascites: treatment and medication. author: Rahil Shah, MD chief editor: Praveen K Roy, updated on May, 04, 2020.
11. Roger Walker and Cate Whittlesea: clinical pharmacy and therapeutics (5th edition) pg no.246.